Cyanide spill threatens health in Hungary

Carl Kovac Budapest

A cyanide spill has rendered Hungary's second longest waterway, the Tisza River, "dead for years to come" and may threaten human health, government officials have warned.

The spill occurred on 30 January at the Aurul precious metals recovery plant near Baia Mare, Romania, when 100 000 m³ of sludge contaminated with cyanide and heavy metals flowed over a 25 m length of dam and down the Szamos and Lapos rivers to the Tisza.

Aurul, an Australian-Romanian joint venture, uses cyanide in a process to recover gold and silver from mine waste material, a practice prohibited in the coun-

tries of the European Union.

Environment officials fear that concentrations of heavy metals—primarily lead, copper, and zinc—in the beds of the affected rivers could work their way into the food chain.

"It is impossible to give an estimate of what the health hazards will be," Miklós Marek, a senior adviser at Hungary's Institute for Environmental Management, said last week. "We need a wider and more detailed investigation."

The Netherlands has already sent experts to the region, and the European Union and the United States have promised technical and financial assistance.

The spill seems to have had little lasting impact on the public water supply along the Szamos and Tisza but has wiped out practically all aquatic life in the rivers. By last week, more than 150 tonnes of dead fish had been pulled from the Tisza.

Fish along one stretch of the



Fisherman Slavko Maric holds a dead perch taken from the polluted Tisza River in Becej

river were found to contain 2.6 mg of cyanide per kilogram of weight. These included carp, cat-fish, and pike—all fish commonly eaten by people in the region. "The Tisza is dead for years to come, and there are several species that are for ever dead," said Gábor Horváth, spokesman

for Hungary's foreign ministry.

The cyanide plume reached the Danube River in Serbia last week. Officials said the cyanide had been sufficiently diluted so as not to pose a major problem to drinking water. However, there was enough to kill fish and micro-organisms in the river.

Cirrhosis may be amenable to telomerase treatment

Abi Berger BMJ

The urgent need for liver transplantation in acute liver failure may be deferred by the findings of researchers in Boston, Massachusetts. Professor Ron DePinho and his team have been looking at ways to prolong the lives of people waiting for donor livers.

They may also have come up with a new treatment for slowing the progression of liver disease (*Science* 2000;287:1253-8).

The progression of liver disease that culminates in cirrhosis is characterised by chronic hepatocyte destruction, with continuing attempts at cell regeneration over a period of years. But a point is reached where the hepatocytes are no longer able to proliferate, and this seems to coincide with the activation of stellate cells in the liver.

Stellate cells are responsible for the production of collagen, which produces bridging fibrosis that is pathognomonic of liver cirrhosis. End stage organ failure is reached.

One hypothesis for why end stage organ disease occurs when it does is that it depends on the length of the cells' telomeres, the genetic caps that protect the ends of chromosomes.

Ron DePinho, professor of medicine and genetics at Harvard Medical School, and colleagues took telomerase deficient mice (genetically modified mice that do not produce telomerase, the enzyme necessary for maintaining telomere integrity) and subjected them to liver ablation. Without the ability to maintain healthy telomeres, these mice could not tolerate the assault on their liver cells, and the fibrous deposition indicative of cirrhosis became apparent.

"We then wondered what would happen if we reintroduced telomerase to these mice," said Professor DePinho.

Using a virus as a vehicle for carrying telomerase into the mouse hepatocytes, Professor DePinho's team discovered that they could indeed block the formation of cirrhosis. This proved a pathogenetic causal link between the shortening of telomeres and cirrhosis. It also suggested to Professor DePinho that diseases such as cirrhosis may be amenable to telomerase treatment.

Painkillers "may need to be sex specific"

Nigel Hawkes Washington

People who constantly complain about their agonising pain are probably telling the truth, according to Professor Jeffrey Mogil of the University of Illinois. He told the American Association for the Advancement of Science in Washington DC last weekend that his work with inbred strains of mice had convinced him that huge differences in the perception of pain are real, not just evidence of a lack of moral backbone.

"There really are differences in our responses to pain," he said. "This knowledge should lead to a destigmatisation of people who are pain sensitive." Professor Mogil spoke at a session devoted to new approaches to the control of chronic pain. Introducing it, Professor Allan Basbaum of the University of California in San Francisco said that recent work had shown that the pain system was rich in unique molecules not found elsewhere in the brain, providing "incredible numbers of new therapeutic targets for

the relief of persistent pain."

One finding reported by Professor Mogil is that-in rodents at least-males and females have slightly different paths for the perception of pain. It has been known for some time that women perceive pain more intensely than men, although the differences are not great and not all studies have confirmed them. Professor Mogil's work suggests that there are qualitative as well as quantitative differences between the sexes.

As well as suggesting that drug companies may one day need to market sex specific painkillers, the discovery illustrates the enormous variety in pain perception among different strains of inbred mice. Some are virtually indifferent to pain, while others are hypersensitive.

Correction

Women taking combination HRT are at greater risk of breast cancer. Owing to an error in the editing of this article (5 February, p 333), it was stated that women in the trial on hormone replacement therapy received oestrogen and progesterone, when it should have said that they received oestrogen and progestogen and progestogen. We apologise for this error.